EFFECT OF INTRAPARTUM AMNIOINFUSION IN MECONIUM STAINED AMNIOTIC FLUID AND PERINATAL OUTCOME

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CERTIFICATE

This is to certify that the dissertation entitled "'**THE EFFECT OF INTRAPARTUM AMNIOINFUSION IN MECONIUM STAINED AMNIOTICFLUID AND PERINATAL OUTCOME'"** is the bonafide original work of **Dr. T. KAVITHA** in partial fulfilment of the requirements for **M.D. Branch – II (Obstetrics and Gynaecology)** Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in March 2008. The period of study was from September 2006 to August 2007.

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DECLARATION

I solemnly declare that the dissertation titled '**THE EFFECT OF INTRAPARTUM AMNIOINFUSION IN MECONIUM STAINED AMNIOTICFLUID AND PERINATAL OUTCOME**' was done by me, Dr.T. Kavitha at Govt R.S.R.M Lying in Hospital attached to Govt. Stanley Medical College, Chennai, under the guidance and supervision of **Prof. Dr. AMRITA PRESCILLA NALINI, MD, DGO.,** Superintendent i/c and Head of the Department, Govt. R.S.R.M. Lying-in-Hospital, Stanley Medical College, Chennai. This dissertation is submitted to Tamilnadu Dr MGR Medical University, Chennai towards the partial fulfillment of requirements for the award of MD Degree in Obstetrics and Gynaecology.

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CONTENTS

INTRODUCTION

Amniotic fluid serves several functions, such as providing a medium for fetal growth and development, protecting the fetus from external trauma, and maintaining intrauterine temperature.

Meconium stained amniotic fluid occurs in 12-20 % of all pregnancies. In the past it was considered as a sign of fetal distress occurring only in response to hypoxia.

But in most cases, meconium in amniotic fluid is benign and associated with term pregnancies. While there is a general agreement that the presence of meconium stained amniotic fluid is associated with increase in perinatal mortality and morbidity, many investigators do not believe that presence of meconium predicts a poor outcome unless it is accompanied by other signs of fetal distress

However, if aspirated by the fetus before or during birth, meconium can obstruct the airways, interfere with gas transfer and cause respiratory difficulties. The increased perinatal mortality and morbidity in the presence of meconium stained amniotic fluid is mainly due to meconium aspiration syndrome, which occurs in 2-10% of these neonates. 50-60% of infants whose amniotic fluid was meconium

stained was reported to have meconium in their tracheas. Treatment strategies of deep suctioning of hypo pharynx and nares during delivery and immediate tracheal suctioning after birth have been successful in reducing meconium aspiration syndrome. But despite aggressive airway clearing, meconium aspiration syndrome is not always prevented, the causative factor being in utero aspiration.

Amnioinfusion dilutes the amniotic fluid and has been studied as an additional tool to prevent meconium aspiration. It helps by physical dilution of the thick meconium and thereby decreasing the toxic effects of aspiration of thick meconium. It also increases the fluid around the fetus and thereby decreases cord compression and hence fetal hypoxia when present.

AIM OF THE STUDY

Evaluation of intrapartum transcervical amnioinfusion as a therapeutic procedure for moderate and thick meconium stained amniotic fluid and its impact on

1. The incidence of intrapartum foetal heart abnormalities.
2. The incidence of intrapartum operative interventions.
3. Neonatal outcome by Apgar score at 1 minute and 5 minutes.
4. Incidence of meconium below the vocal cords by direct laryngoscopy.
5. Incidence of meconium aspiration syndrome.

REVIEW OF LITERATURE

In 1976 Gabbe[3] and coworkers demonstrated in a rhesus monkey that removal of amniotic fluid produced variable deceleration and the replenishment of fluid with saline relieved the deceleration.

In 1983 Miyazaki and Taylor[6] infused saline through intra uterine pressure catheter to women in labour who had either variable deceleration or prolonged deceleration attributed to cord entrapment. They found that this improved heart rate pattern in half the patients.

In 1985 Miyazaki and Nevarez[7] randomized 96 pregnancies and found multiparous women in labour with cord compression pattern who were treated with amnioinfusion less often-required caesarean delivery for foetal distress.

In 1978 Meis *et al*[5] evaluated patients with meconium passage in early and late labour and graded the presence of meconium as light or heavy. Heavy meconium passage in early labour was associated with increased foetal morbidity including abnormal foetal heart pattern, prolonged second stage of labour and increased operative interventions. It was also associated with higher incidence of neonatal morbidity and mortality.

Carson *et al*[4] 1976 described combined obstetric and pediatric approach to infants tracheal suctioning at delivery.

Wenstrom *et al*[8] 1989 randomly compared 85 patients with thick meconium. The study group was infused with 1000ml normal saline solution initially and every 6 hours until delivery. Control patients received routine management .The incidence of low 1 minute Apgar score, meconium below the vocal cords and operative delivery were significantly less in patients receiving amnioinfusion. They noted that amnioinfusion is a simple inexpensive and safe technique that reduces the incidence of meconium below cords and improves the obstetric outcome in patients.

Sadovsky *et al*[9]1989 demonstrated that 19 cases of labour treated with amnioinfusion for thick meconium had a better neonatal outcome compared with 21 cases treated routinely. The frequency of neonatal acidemia and positive pressure ventilation was significantly reduced in the infused group.

In 1990 Posner MD *et al*[10]. did a study on the effect of amnioinfusion on uterine pressure and activity and reported that a significant increase in uterine tone was noted during and after the infusion.

In 1992 Charles J *et al*[14] studied 170 term and post term patients with thick meconium chosen to receive amnioinfusion or standard obstetric care without amnioinfusion. The rate of foetal distress,caesarean section for foetal distress and rate of meconium aspiration syndrome were significantly reduced with amnioinfusion.

Eriksson and colleagues[22] in 1994 found that amnioinfusion in pregnancies complicated by thick meconium stained amniotic fluid reduced the risk of meconium below the cords and respiratory distress in newborn.

Lo *et al*[20] in 1993 and Cialone *et al*[23] in 1994 reported a reduced frequency of acidemia, meconium aspiration syndrome and other neonatal complications in conjunction with amnioinfusion in the setting of thick meconium.

In 1996 Ou Zounian JG *et al*[28] did retrospective study to evaluate the use of intrapartum amnioinfusion in women undergoing a trial of labour after a previous cesarean delivery and concluded that amnioinfusion appears to be an acceptable procedure in women with previous cesarean births.

Hofmeyer *et a/*[34] 1998 for the Collaborative Randomized Amnioinfusion for Meconium Project (CRAMP1) South Africa did a multicentre randomized controlled trial and found that amnioinfusion for meconium stained amniotic fluid reduces the incidence of meconium aspiration syndrome.

Mahomed *et a/*[35] 1998 (CRAMP 2) Zimbabwe found that meconium aspiration syndrome was significantly less frequent in amnioinfusion group and there was trend towards fewer perinatal deaths. In the same study there was no difference in the rate of caesarean section.

In 1999 Edwards RK *et al*[37] did randomized study about prophylactic cefazolin in amnioinfusion administrate for meconium stained amniotic fluid and concluded that prophylactic use of cefazoline in Amnioinfusion did not significantly reduce the rates

of maternal or neonatal infection in patients with meconium stained amniotic fluid.

John Pierce *et al*[39] 2000 did a Meta analysis of 13 prospective clinical trials of intrapartum amnioinfusion for meconium stained fluid. They reached a conclusion that amnioinfusion significantly reduced the frequency of meconium aspiration syndrome, meconium below the vocal cords and neonatal acidemia. The overall caesarean delivery rate was also significantly lower without increased postpartum endometritis.

In 2000 Ask AK[40] said amnioinfusion can be an effective preventive measure for managing patients with meconium stained amniotic fluid.

Vinita Das *et al*[41] did a prospective case control study in 2001 regarding the safety and efficacy of amnioinfusion during labour complicated by meconium stained liquor and found that amnioinfusion decreases the cesarean rate, incidence of meconium aspiration syndrome and perinatal mortality.

In 2001 Wiswill TE[42] proposed that antenatal therapies should include amnioinfusion, intrapartum maneuvers should include oropharyngeal suctioning prior to delivery of the baby's shoulders, the postnatal intervention of intubation for intratracheal suctioning should be reserved for the non-vigorous meconium stained infant.

In 2001, Puertas A, *et al*[43] did randomized trial of prophylactic amnioinfusion and concluded that amnioinfusion improves the neonatal outcome and reduces the frequency of caesarean sections.

A.M Rathor *et al*[44] in 2002 did a randomized controlled trial in 200 women

and found that caesarean section rate in amnioinfusion group was less than control group and amnioinfusion was associated with significant decrease in the incidence of meconium at vocal cords, improvement in one minute Apgar score, respiratory distress and fewer admissions to NICU compared with that of controls.

In 2002 Gonzalez JL *et al*[45] did prospective randomized study using normal saline or ringer lactate solution for amnio infusion in women with thick meconium in the amniotic fluid. Cord blood arterial sampling was analysed for sodium, potassium and chloride plasma concentrations and PH. There was no significant difference in cord blood arterial plasma concentrations of Sodium, Potassium, Chloride and PH.

Sood M[46] Charulatha, Dimple, Aggarval N, Faridi MM, did randomized controlled trial in 2004 regarding amnioinfusion in thick meconium and found that amnioinfusion resulted in relief of deceleration and significant reduction in the incidence of meconium below the vocal cord and concluded that transcervical intrapartum amnioinfusion is a safe, simple and inexpensive technique that reduces operative intervention and improves neonatal outcome, and is of tremendous value in developing countries.

In 2004, Ashfaq F[47], Shah AA did controlled clinical trial of effect of amnioinfusion for meconium stained amniotic fluid on perinatal outcome and found that the amnioinfusion significantly decreased the cesarean rate, perinatal mortality and morbidity, meconium aspiration syndrome, neonatal admission in NICU.

In 2006, Parth Mukhopadhyay[51], Tapan Naskar, Rabindranath Dalui, did a study about role of intrapartum amnioinfusion in meconium stained amniotic fluid and found that amnioinfusion decreases the incidence of cesarean rate owing to fetal distress, decreases the meconium in the treachea, reduces the incidence of to meconium aspiration syndrome and birth asphyxia.

In 2006, ACOG Committee[50] Obstetric practice said that routine prophylactic amnioinfusion for the dilution of amnioinfusion for meconium stained amniotic fluid should be done only in the setting of additional clinical trials. However, amnioinfusion remains a reasonable approach in the treatment of repetitive variable decelerations, regardless of amniotic fluid meconium status.

Cochrane database[48] 2006 says amnioinfusion is associated with a reduction in heavy meconium staining of liquor, variable fetal heart deceleration and reduced caesarean section. Also associated with reduction in meconium aspiration syndrome, neonatal hypoxic ischaemic encephalopathy and neonatal ventilation or NICU admission. The database also showed a trend towards reduced perinatal mortality. It concluded that amnioinfusion is associated with improvement in perinatal outcome particularly in settings where facilities for perinatal surveillance are limited.

In 2006, Velaphi[49] S. Vidyasagar D provides evidence based recommendations regarding the benefits of amnioinfusion prior to delivery, oral suctioning of the new born prior to delivery of the shoulder and the practice of routine endotrachaeal suctioning of the newborn born through meconium stained

amniotic fluid in preventing meconium aspiration syndrome.

In 2007 Das AK,[52] Jana N, Das Gupta S, Samanta B did prospective comparative study about the effect of amnioinfusion in meconium stained amniotic fluid and found that amnio infusion significantly reduced the incidence of low APGAR score, meconium aspiration syndrome, incidence of cesarean deliveries and perinatal death.

In 2007, XUH, Hofmeyr J *et al*[53] did randomized trials comparing amnioinfusion with no amnioinfusion for women in labour with meconium stained amniotic fluid and found that in clinical settings with standard peripartum surveillance the evidence does not support the use of amnio infusion for meconium stained amniotic fluid. In settings with limited peripartum surveillance, when complications of meconium stained amniotic fluid are common, amnioinfusion appears to reduce the risk of meconium aspiration syndrome.

Amnioinfusion is technically feasible even in developing countries with limited intrapartum facility.

AMNIOINFUSION-GENERAL CONSIDERATIONS

Definition

Amnioinfusion is a procedure in which a physiologic solution [such as normal saline] is infused into the amniotic cavity. There are three predominant indications[56] for which amnioinfusion has been used:

1) Intrapartum amnioinfusion for meconium stained amniotic fluid.

2) Treatment of variable or prolonged decelerations.

3) Amnioinfusion for oligohydramnios.

AMNIOINFUSION FOR OLIGOHYDRAMNIOS

Transabdominal amnioinfusion has been attempted for diagnostic purposes in women with second or third trimester oligohydramnios.

Instillation of saline will improve the sonological visualization[16] and more accurate diagnosis of congenital anomalies or IUGR in case of oligohydramnios.

In addition to this, repeated trans abdominal amnioinfusions in patients with normally grown fetus but persistent oligohydramnios would prevent pulmonary hypoplasia and compression defects[12].

INTRAPARTUM AMNIOINFUSION FOR VARIABLE DECELERATIONS

Many reports[24] have documented relief of variable decelerations with trans vaginal amnioinfusion. Presumably more fluid is associated with less cord compression and thus relieves the variable decelerations, which occur due to cord compression[6].

INTRAPARTUM AMNIOINFUSION FOR MODERATE OR THICK MECONIUM STAINED AMNIOTIC FLUID

Multiple randomized studies[23,34,35] have reported that amnioinfusion for moderate or thick meconium stained amniotic fluid is associated with a significant decrease in meconium aspiration.

For amnioinfusion either Ringer lactate or Normal saline can be used, the type of solution used has not shown to affect the outcome. These solutions do not cause any electrolyte imbalance in the fetus[45].

Glantz and Letteney[30] compared using warmed [37^0C] versus room temperature infusions; they found no difference in the outcome. They also compared using infusion pump versus gravity and found both to be equally effective.

Several protocols have been described for amnioinfusion:

1. An initial bolus of 500-600ml in 30-60 minutes followed by a maintenance rate of 3ml/minute till delivery.
2. An initial bolus of 500-600ml in 30-60 minutes. Repeat the dose after 4 hours if delivery does not occur.

The protocol used in this study was to infuse 500ml of normal saline as initial bolus dose over 30 minutes, followed by 500ml more at a rate of 2-3 ml/minute. The use of maximum 1 liter was considered safe due to lack of accurate intrauterine pressure monitoring or cardiotocographic monitoring[41].

RISKS AND COMPLICATIONS OF AMNIOINFUSION

Reported complications include uterine hypertonus, fetal heart rate abnormalities, chorioamnionitis, uterine rupture, placental abruption and maternal cardiac and respiratory failure[24]

Maher *et al*[25] reported two cases of amniotic fluid embolism in women when an amnioinfusion by infusion pump was ongoing. Several authors have reported the occurrence of excessive uterine contractions or unusually rapid progress of labour associated with amnioinfusion.

There is a hypothesis that excessive uterine contractions in some cases are related to extra amniotic placement of amnioinfusion catheter with simultaneous prostaglandin release[38] Rapid absorption of the infused fluid could account for the

reported cases of pulmonary edema. Consensus of reported data suggest that there is no increased risk of maternal and neonatal infection with amnioinfusion. Owen *et al*[11] noted decrease in endometritis in women with amnioinfusion compared with those in control.

NEONATAL CARE FOLLOWING AMNIOINFUSION

Assuming that most of the meconium aspiration occurs at first neonatal breath, Carson[4] and associates (1976) reported that suction of fetal nasopharynx immediately after delivery of the head but before the delivery of the shoulders combined with tracheal intubation and suction was effective in reducing the incidence and severity of meconium aspiration.

But Suresh and Sarkar (1994) noted that despite careful suctioning and absence of neonatal breathing before intubation, 75% still had meconium at trachea.

The current view is that meconium aspiration occurs due to fetal breathing movements, causing inhalation of amniotic fluid with meconium. The breathing movements, which cause inhalation of amniotic fluid, are of two types: gasping and deep breathing[54].

Gasping is a normal response to hypoxemia and can be induced experimentally by occluding the umbilical cord or by occluding the maternal aorta.

The fetus may also inhale meconium by deep irregular breathing in utero, not initiated by hypoxia. These breaths become more frequent as gestation advances, and comprise 10% of all fetal breathing movements. Fetal hypercapnoea and acidemia also increase these breathing movements but they still occur in most if not all normal fetuses[54].

Thick meconium is viscid enough to obstruct neonatal airway and concentrated enough to cause significant chemical injury to tissues. Starks (1980) found that thick meconium was associated with a higher percentage of low Apgar scores. Mahomed and colleagues (1994) reported that patients with thin meconium had outcomes indistinguishable from those with clear fluid.

Amnioinfusion dilutes the meconium and this reduces the toxic effects due to aspiration of thick meconium. It also increases the volume of fluid around the fetus, thereby decreasing the possibility of fetal distress related to cord compression[6]

SIGNIFICANCE OF MECONIUM PASSAGE IN UTERO

Meconium, an odorless thick blackish green material is first demonstrable in the fetal intestine during the third month of gestation. It is an accumulation of debris that consists of desquamated cells from the alimentary tract and skin, lanugo hairs, fatty material from vernix caseosa, amniotic fluid, and various intestinal secretions.

The quantity of meconium in fetal gut is small during the first two trimesters, but increases rapidly during the third. Because the internal and external anal sphincters are usually closed during fetal life, amniotic fluid ordinarily remains clear. Various stimuli however are known to cause relaxation of sphincter tone and subsequent passage of meconium into the amniotic fluid.

It is widely believed that any insult causing hypoxia in fetus will cause fetal hyper peristalsis and relaxation of the fetal anal sphincter[55]. However as Hon suggested parasympathetic stimulation from cord compression may stimulate meconium passage without concomitant hypoxia.

It is also possible that meconium is passed as a result of spontaneous gastro intestinal motility that reflects physiologic maturation of fetal gut. As the vast majority of fetuses have no acid base abnormality, the presence of meconium itself is not a sensitive or specific indicator for fetal compromise[54].

A number of studies have failed to show any consistent effects of meconium stained amniotic fluid on Apgar scores, fetal scalp pH or incidence of

fetal heart rate abnormalities. (Abramnici et al 1974; Miller *et al* 1975; Baker *et al* 1992). Moreover one study found that although meconium stained amniotic fluid correlated poorly with markers of acute intrauterine asphyxia, (pH, lactate, hypoxanthine concentration) it correlated well with blood erythropoietin concentration (marker of chronic asphyxia)[55] .

Despite these theories, most obstetricians agree that meconium stained amniotic fluid in connection with fetal heart rate abnormalities is a marker for fetal distress and is associated with increased perinatal morbidity and mortality.

MECONIUM ASPIRATION SYNDROME

Whatever be the stimulus, once meconium has been passed, any episode, either fetal gasping or respiratory attempts by the foetus can cause aspiration of amniotic fluid containing meconium into the fetal tracheo bronchial tree. Meconium aspiration thus occurring can obstruct the airway; interfere with gas exchange and cause severe respiratory distress.

Meconium aspiration syndrome can occur in 10% of cases with meconium stained amniotic fluid. It comprises a significant range of respiratory compromise. In its mildest form[55], the disease may present with neonatal tachypnoea associated with normal pH and lower pCO2, which resolves within 2-3 days. Clinically this mild respiratory morbidity may be indistinguishable from transient tachypnoea of newborn. In the more severe form, the syndrome can present as severe hypoxemia, acidosis, pnuemothorax and respiratory failure a

few hours after birth. Pulmonary arterial vasospasm may lead to right to left shunting through patent foramen ovale or ductus arteriosus. Hypoxia further stimulates pulmonary hypertension and the downward spiral continues ultimately leading to convulsions, renal failure, disseminated intravascular coagulation and heart failure.

Clinically the infants with meconium aspiration syndrome can have evidence of over inflation of lungs, with a barrel chest. Auscultation can reveal diffuse crepitations and rhonchi. The chest radiograph shows patchy areas of atelectasis and areas of over inflation. Pnuemothorax and pnuemomediastinum are common (10-20% Peters and Pendleton 1989). Pleural effusion may be present in about 30%. But the severity of chest x-ray abnormalities may not correlate with the severity of clinical disease.

PATHOPHYSIOLOGY

The pathophysiology of meconium aspiration is extremely complex due to interplay of a large number of mechanisms. Perinatal asphyxia is a critical underlying factor in the pathogenesis of meconium aspiration syndrome. The pulmonary abnormalities in meconium aspiration syndrome are related primarily to acute airway obstruction, with obstructive emphysema due to ball valve effect.

Due to the direct irritation and toxicity of meconium constituents, there is a marked alveolar and parenchymal inflammation and edema with leakage of proteins into the airways. There is release of inflammatory mediators like

cytokines. Meconium adversely affects the neutrophil function by inhibiting phagocytosis with increased risk of infections. Surfactant dysfunction may occur due to cytotoxic effect on type 2 pnuemocytes and decreased level of surfactant proteins A and B. there is increased airway resistance and reduced compliance of lungs.

Pulmonary vasoconstriction occurs due to injury of the vascular bed of the lung. In these infants, vascular smooth muscle extends into the walls of normally non-muscular intra acinar arterioles and reduces their luminal diameter, which subsequently interferes with normal postnatal drop in pulmonary vascular resistance. In addition these infants may demonstrate plugs of platelets in their small vessels which reduces the cross sectional area of the pulmonary vascular bed.

MANAGEMENT OF INFANT DELIVERED THROUGH MECONIUM STAINED AMNIOTIC FLUID

In all cases of meconium stained amniotic fluid, obstetrician should do the suction of oropharynx, mouth and nose as soon as the head of the baby is delivered.

In 1974, a prospective study by Gregory *et al*[1] recommended laryngoscopy and tracheal intubation with suctioning for all newborns born through thick particulate meconium. In 1976 Carson *et al*[4] reported a decrease in meconium aspiration syndrome with suctioning of hypopharynx before the

shoulders were delivered and before the first postnatal breath. They recommended routine tracheal suctioning after this was not necessary unless meconium was visible at vocal cords or hypopharynx. Many studies (Wisewell *et al*[17] Hageman *et el;* Strickland *et al)* have shown beneficial effects of aggressive airway management.

In contrast AAP[18] guidelines of perinatal care recommends endo tracheal intubation and direct tracheal suction only if there is meconium at the larynx on laryngoscopy and the infant is depressed.

But the study by Wiswell[17] showed that as many as 41-54% of infants in whom meconium aspiration syndrome developed had Apgar score of 8 or greater. Thus a vigorous baby can still develop meconium aspiration syndrome.

So the current management is for any infant delivered with thick meconium stained amniotic fluid: place in radiant warmer, immediate intubation and suction of trachea via ETT with meconium aspirator, repeat the suction till clear, assess for respiratory distress, and empty the stomach contents[18]

Meconium Stained Fluid Detected[18]

↓

Notify person skilled in neonatal resuscitation and capable of ET intubation available

↓

Suction mouth and nasopharynx prior to delivery of the shoulders

Place infant on radiant warmer in Trendelenburg

↓

Thick or Particulate Meconium

Immediately intubate and suction trachea via ETT with meconium aspirator.

Repeat until clear

↓

Assess for respiratory distress and

Further management for MAS.

Empty stomach contents

MANAGEMENT OF MECONIUM ASPIRATION SYNDROME

Symptomatic infants with meconium suction from the trachea should be given chest physiotherapy and warm humidified oxygen to breathe. Due to high incidents of air leaks, positive pressure of ventilation must be avoided if possible. Mechanical ventilators should be used for infants with apnoea from birth asphyxia or for those infants who cannot maintain their PaO2 >50mm Hg in 100% Oxygen[55].

As meconium aspiration pneumonia is an obstructive lung disease the time constant for expiration is prolonged in severely involved areas of lungs. Careful attention must be given to expiratory type to prevent inadvertent PEEP, further gas trapping and alveolar rupture.

Meconium enhances bacterial growth by reducing the host resistance[55]. But no studies confirm that infection plays a role in the pathogenesis of meconium aspiration syndrome. Since there is difficulty indistinguishing meconium aspiration syndrome from bacterial pneumonitis, many routinely treat infants with having meconium aspirations with antibiotics.

The use of corticosteroids for treating meconium aspiration syndrome is not recommended. The time taken for weaning to room air is prolonged with the use of steroids[55]

As discussed earlier there is evidence that infants with meconium aspiration syndrome have surfactant inactivation. Trials have found that surfactant replacement therapy if started within six hours of birth improve oxygenation, reduces air leaks, reduces pulmonary morbidity and shortens hospital duration of stay. (Findly *et al* I996).

MATERIALS AND METHODS

The Study was conducted at Govt RSRM Lying-in Hospital from September 2006 to August 2007. For this prospective randomized controlled clinical trial, participants were 200 women in labour at term with moderate or thick meconium stained amniotic fluid.

Inclusion Criteria

1) Singleton pregnancy
2) Vertex presentation
3) Gestational age between 37 and 41 weeks
4) Normal fetal heart rate with regular rhythm.
5) Having moderate or thick meconium stained amniotic fluid on spontaneous rupture of membranes or after doing artificial rupture of membranes in labour ward.

Exclusion criteria

1) Fetal malpresentations
2) Multiple gestations.
3) Fetal congenital anomalies.
4) Prelabour rupture of membranes.
5) Polyhydramnios.
6) Chorioamnionitis.
7) Antepartum hemorrhage.
8) Medical diseases complicating pregnancy.

Procedure

Each patient was carefully examined to rule out any risk factor, confirmation of gestational age was done by detailed menstrual history, clinical examination and previous scan report if the patient had taken already.

Abdominal examination was done and fetal presentation determined. Duration and frequency of uterine contractions were assessed. The fetal heart rate was auscultated with Pinard Foetoscope.

Vaginal examination was done to assess the stage of labour and to confirm the presentation. The amniotic fluid was examined after spontaneous or artificial rupture of membranes and graded as follows :

Grade I thin meconium: having uniform greenish staining and watery.

Grade II moderate: having thicker greenish staining but still watery.

GradeIII thick meconium: Green with particulate matter having pea soup quality. [Thick tenacious and opaque]

These patients were categorized into amnioinfusion and no amnioinfusion group in a 1: 1 ratio i.e. the first patient was given amnioinfusion, whereas the second patient was taken as control group [without amnioinfusion]. Procedure was explained to the patient and consent obtained.

In the amnioinfusion group patient was asked to lie in dorsal position and an autoclaved rubber catheter was introduced transcervically under aseptic precautions so that its tip lies just above the baby's head. Normal saline at room temperature was infused through the catheter using intravenous tubing.

The amnioinfusion started with an initial bolus of 500 ml normal saline infused over 30 minutes. Then 500 ml more was infused at a rate of 30-45 drops [2-3ml] per minute. The use of maximum one litre was considered safe. The control group received no catheter placement. Both groups were then managed in labour ward according to routine policy.

Uterine tone and frequency of contractions were assessed by abdominal palpation. Oxytocin drip was started if contractions were not effective. Fetal heart rate was auscultated every 15 minutes during the first stage of labour and every 5 minutes during the second stage using Pinard foetoscope. Fetal distress was defined as fetal bradycardia [<120 beats / minute] or fetal tachycardia [>160 beats / minute].

Progress of the labour was monitored and obstetric assistance was given whenever necessary. Forceps application or delivery by caesarean section was done for fetal heart rate abnormalities or failure to progress such as arrest disorders or protraction disorders as indicated.

During delivery as soon as baby's head was delivered, the eyes were cleaned and suction of oropharynx, mouth and nose was done before the delivery of the shoulders by the obstetrician. Any mucus or meconium was aspirated. Pediatric resident postgraduate was called to attend the new born. After suctioning of the oropharynx and nasopharynx laryngoscopic visualization of the vocal cords was done by the pediatrician. The presence or absence of meconium below the vocal cords were evaluated and documented.

The staining of baby with meconium and 1 minute and 5 minute Apgar scores were observed and recorded. If the baby had respiratory distress or 5minute Apgar score less than 6 then baby was taken to neonatal unit and treated accordingly.

After delivery patients were observed in labour ward for two hours and then shifted to postnatal ward. Any sign of puerperal pyrexia was watched for in the mother.

Neonatal and maternal well being was monitored till the day of discharge from the hospital.

ANALYSIS OF STUDY OBSERVATION

TABLE-1

AGE IN YEARS

Age	Amnioinfusion	Control
18-20	24	28
21-29	70	67
>30	6	5

P = 0.79 NS

In this study the age of the amnioinfusion group was matching with the control group.

TABLE-2

PARITY

Parity	Amnioinfusion	Control
Nullipara	65	61
Paral	28	30
Para2	7	9

In this study parity of both the groups was matching. In both groups nulliparas were predominant and contributed to 65% of the study group and 61% of the control group

TABLE-3

GESTATIONAL AGE

Gestational Age	Amnioinfusion	Control
37-40 Weeks	65	69
40-41 Weeks	35	31

P=0.55 NS

The sample groups of amnioinfusion and control were matching in relation to their gestational age. Most of the patients were between 37-40 weeks of gestational age (65 in amnioinfusion and 69 in control group)

TABLE-4

PHASE OF LABOUR ON INCLUSION

Phase	Amnioinfusion	Control
Latent	36	31
Active	64	69

P=0.45NS

In the amnioinfusion group 36 patients were in the latent phase of labour compared to 31 in the control group. In the control group 69 patients were in the active phase of labour compared to 64 in the amnioinfusion group

TABLE-5

LABOUR CHARACTERISTICS

Oxytocin Augmentation	Amnioinfusion (n=100)	Control (n=100)
Present	37	31
Absent	63	69
Rupture of Membranes to delivery Interval (hrs)	1.73	1.56

Oxytocin augmentation was required in 37 patients in the amnioinfusion group and 31 patients in the control group. (P= 0.37 P value >.05 NS). The mean interval from rupture of membranes to delivery was 1.73hours in the amnioinfusion group and 1.56 hours in the control group. This shows that there is no significant difference in the use of oxytocin for augmentation or in the duration of labour with amnioinfusion.

TABLE-6

INTRAPARTUM FOETAL HEART RATE ABNORMALITIES

Bradycardia, Tachycardia	Amnioinfusion n=100	Control n=100
Present	28	42
Absent	72	58

P=0.04S

There were more cases of fetal distress in the control group (42%) compared with the amnioinfusion group (28%). P value is <. 05 This shows that amnioinfusion causes a significant reduction in the fetal heart rate abnormalities

during labour.

TABLE-7

MODE OF DELIVERY

Mode	Nullipara		Para l		Para 2		Total	
	Amnio Infusion	Control	Amnio Infusion	Control	Amnio Infusion	Control	Amnio Infusion	Control
Labour Natural	28	27	23	19	4	8	55	54
Outlet Forceps	4	5	-	-	-	1	4	6
LMC forceps	1	1	1	-	2	-	4	1
LSCS	32	28	4	11	1	-	37	39

When mode of delivery is considered 55 in the amnioinfusion group and 54 in the control group delivered by labour natural. 4 patients in amnioinfusion group and 6 patient in the control group had outlet forceps delivery. Low midcavity forceps was used in 4 patients belonging to the amnioinfusion group and 1 patient in the control group. 37 patients in the amnioinfusion group and 39 patients in the control group were delivered by LSCS. (P value >.05). This shows that amnioinfusion is not influencing the mode of delivery in this study

TABLE-8
RELATIONSHIP OF PHASE OF LABOUR TO MODE OF DELIVERY

Mode	Amnioinfusion				Control			
	Latent: n=36		Active: n=64		Latent: n=31		Active: n=69	
	Number	%	Number	%	Number	%	Number	%
Labour natural	6	16. 67 %	49	76.56%	6	19.35%	48	69.57%
Forceps	--	--	8	12.5%	-	-	7	10.14%
LSCS	30	83.33%	7	10.94%	25	80.65%	14	20.29%

Out of the 36 patients included in the amnioinfusion group in the latent phase, only 6(16.67%) delivered by labour natural. No patient had forceps delivery, and caesarean was done for the majority of patients 30 (83.33%). In the control group there were 31 patients in the latent phase of labour. Out of these 6(19.35%) delivered by labour natural and cesarean was done for the majority 25(80.65%).

In the amnioinfusion group 64 patients were in the active phase and 49 (76.56%) of them delivered by labour natural. Forceps was applied for 8(12.5%) patients and only 7(10.14%) patients were taken for cesarean section. Out of the 69 patients in the control group in the active phase of labour 48 (69.57%) delivered by labour natural, forceps was used for 7(10.14%) patients and cesarean was done only in 14(20.29%) patients.

This shows that most of the patients with meconium stained amniotic fluid in the active phase of labour delivered by labour natural irrespective of whether amnioinfusion was given or not. And the patients that were included in the latent phase cesarean was the commonest mode of delivery in both the groups.

TABLE-9

INTRAPARTUM OPERATIVE INTERVENTION

	Amnioinfusion (n=100)	**Control (n=100)**
Forceps	8	7
LSCS	37	39
Total	45	46

P= 0.94NS

Regarding the number of intrapartum operative interventions, 45 in amnioinfusion group and 46 in the control group (P>.05) no significant decrease was seen with the use of amnioinfusion. When the incidence of LSCS alone was taken into consideration, it was 2% less in the amnioinfusion group. But this difference is not statistically significant.

TABLE 10

REASON FOR OPERATIVE INTERVENTION

Reason		**Amnioinfusion**	**Control**
Foetal Distress	LSCS	18	31
	Forceps	2	4
	Total	20	35
Other	LSCS	19	8
	Forceps	6	3
	Total	25	11

P=0.05S

When the reason for operative intervention was considered there was more number of cesareans for fetal distress in the control group than in the

amnioinfusion group (31 vs. 18 P<. 05). This difference is significant. The total number of operative interventions for fetal distress was 20 in the amnioinfusion group whereas it was 35 in the control group (P<. 05). These data shows that amnioinfusion decreases the incidence of operative interventions for fetal distress.

TABLE-11

NEONATAL APGAR SCORE

Apgar score		**Amnioinfusion**	**Control**
1min	0-7	41	60
	8-10	59	40
5min	0-7	13	24
	8-10	87	76

1 minute Apgar score <7 is seen in 41 infants in the amnioinfusion group and 60 infants in the control group. This difference is significant P value <. 05 .13 infants belonging to the amnioinfusion group and 24 infants in the control group had <7 5 minute Apgar. This difference was also found to be significant with a P value of <. 05 Thus amnioinfusion has improved the Apgar of neonates at 1 minute and at 5 minutes

TABLE-12

BIRTH WEIGHT OF BABIES

Weight in Kgs.	Amnioinfusion	Control
2-2.5	30	29
2.6-3.0	53	50
>3	17	21
Mean	2.77	2.78

P=0.77NS

The mean birth weight of the babies in the amnioinfusion and control group is almost equal 2.77 and 2.78.

TABLE 13

MECONIUM BELOW VOCAL CORDS AND MECONIUM ASPIRATION SYNDROME

Meconium below vocal cords	Amnioinfusion			Control		
	Number n=100	MAS		Number n=100	MAS	
		No.	%		No.	%
Present	20	4	20	36	15	41
Absent	80	2	2.5	64	3	4.68

Meconium below the vocal cords in direct laryngoscopy was found in 20 neonates from the amnioinfusion group and 36 neonates from the control group. (P= 0.01P value<.05) there is a significant reduction in the number of neonates with meconium below the level of vocal cords in the amnioinfusion group. In the amnioinfusion group 6 neonates developed meconium aspiration syndrome

compared to 18 in the control group. There is a significant reduction in the incidence of meconium aspiration syndrome (P=0.01Pvalue<.05) in the amnioinfusion group Meconium aspiration syndrome developed in 2 (2.5%) of the neonates in the amnioinfusion group who did not have meconium at the level of vocal cords compared to 3(4.68%) in the control group.

TABLE 14

REASONS FOR NEONATAL UNIT ADMISSION

Reason	Amnioinfusion	Control
Meconium smeared baby for observation	9	14
Meconium aspiration syndrome	6	18
Hypoxic ischaemic encephalopathy	1	I
Birth asphyxia	4	5
Total	20	38

20 babies from the amnioinfusion group and 38 babies from the control group were admitted in the neonatal unit. In the control group meconium aspiration syndrome is the predominant indication for admission. There was one baby with hypoxic ischaemic encephalopathy from each group. 4 babies from amnioinfusion group and 5 babies from the control group had birth asphyxia.

TABLE 15.

DURATION OF NEONATAL UNIT ADMISSION

No of days	Amnioinfusion		Control	
	Number n =20	%	Number n=38	%
1-4	14	70%	25	65.79%
5-10	5	25%	11	28.95%
>10	1	5%	2	5.26%
Mean	3.2		3.6	

The mean duration of stay in the neonatal unit was 3.2 days in the amnioinfusion group and 3.6 days in the control group. This difference was not significant statistically.

TABLE 16

PERINATAL MORTALITY RATE AND MORBIDITY RATE

	Amnioinfusion	Control
Mortality	1/100	3/100
Morbidity	20	38

Perinatal mortality was one in the amnioinfusion group and three in the control group. One baby in the amnioinfusion group died after 1 days in the neonatal unit. That baby was diagnosed to have hypoxic ischaemic encephalopathy stage III. At birth itself the baby had low Apgar scores both at 1 minute and 5 minute. The baby also showed the presence of meconium below the vocal cords at birth. Among the control group 3 babies died in the neonatal

period. One baby died due to ischaemic encephalopathy stage III on the Fifth day. Two babies had meconium aspiration syndrome and died on the First day. The perinatal morbidity is significantly reduced in the amnioinfusion group20 versus 38 in the control Group. P Value <.05

TABLE 17

MATERNAL COMPLICATION

Puerperal Pyrexia	**Amnioinfusion (n=100)**	**Control (n=100)**
Present	2	6
Absent	98	94

2 patients in the amnioinfusion group had fever compared to six patients in the control group. There was no significant difference in the maternal complication between the two groups.

DISCUSSION

In this study 100 patients received amnioinfusion and 100 patients did not receive amnioinfusion for moderate and thick meconium stained amniotic fluid.

The amnioinfusion group and control group were matched with respect of age, parity and gestational age. More patients in amnioinfusion group were in the latent phase of labour on inclusion but this difference was not significant.

The number of patients receiving oxytocin for labour augmentation is not influenced by amnioinfusion (37% vs. 31% P value >.05). In the study by Usta *et al*[27] the amnioinfusion group had significantly higher incidences of oxytocin use than the control group (44% vs. 23% P<. 001). The study by Cialone *et al*[23] also showed the patients receiving amnioinfusion had greater oxytocin requirement in labour.

In this study there is no statistically significant difference between the two groups in the rupture of membranes to delivery interval(1.73 hrs vs. 1.56 hrs). This is correlating with the study by Cialone *et al*[23] in which the rupture of membrane to delivery interval was similar in both groups. Usta *et al*[27] showed longer mean rupture to delivery interval in the amnioinfusion group.

The incidence of fetal distress in this study was 28% in the amnioinfusion group and 42% in the control group. (P<. 05) This is in par with Wenstrom[8] and Parsons study which showed decrease incidence of fetal distress in the amnioinfused patient. But in contrast the study by Rogers *et al*[20]showed a higher

incidence of fetal distress in the amnioinfused group. (30.5% vs. 19.7%) Erikson *et al*[22] also demonstrated that the incidence of fetal distress was not significantly different between the two groups in contrast to this study.

In both the groups in this study most of the patients included in the latent phase of labour delivered by caesarean section while most of the patients included in the active phase of labour delivered vaginally

There was no significant difference between the two groups regarding the mode of delivery. Results from other studies on the effect of intrapartum amnioinfusion on cesarean rates are given in table 18.

TABLE 18

INTRAPARTUM AMNIOINFUSION AND CESAREAN RATES

Study	Amnioinfusion	Control
Spong et al 1994	8/43 (18.6)	9/50 (18.0)
Cialone et al 1994	14/47 (29.8)	11/58 (19.0)
Khosla et al 1997	1/25 (4.0)	7/25 (28.0)
Hofmeyr et al 1998	70/167 (41.9)	68/159 (42.8)
Mahomed et al 1998	30/317 (9.5)	37/328 (11.3)
Moodley et al 1998	3/30 (10.0)	7/30 (23.0)
A.M Rathor et al 2002	21/100 (21.0)	36/100 (36.0)
Parthe Mukhopadhyay et al 2006	18/93 (19.3)	39/93 (41.5)
Das A.K. et al 2007	18/100 (18)	30/100 (30)

The last three studies mentioned in table 18-show reduction in the cesarean section rate with amnioinfusion while other studies do not show a significant difference.

In this study the number of operative deliveries for fetal distress was significantly lower than that in control group (18% vs.31% P<. 05). This is similar to the result in the study by A.M.Rathore *et al*[44,] (12% vs. 26%). Parthe Mukhopadhyay *et al*[51] (19.3% vs. 41.5%). Lo and Rogers[20], Moodley *et al*[36] and Marci *et al*[19] also reported similar findings. Eriksen *et al*[22] and Sponge et *al*[26] reported no effect on caesarean section for fetal distress. Usta *et al* [27] reported a higher incidence of caesarean delivery in the amnioinfusion group. (28% vs. 17%) especially for fetal distress (16% vs. 11%) The CRAMP 1 and 2[34,35] also showed no significant difference in the caesarean section rates for fetal distress between the two groups.

In this study the 1-minute Apgar rate of < 7(41% vs.60% P<.05) and 5-minute Apgar rate of <7 (13% vs.24% P<.05) was significantly lower in the amnioinfusion group. Parthe Mukhopadhyay *et al*[51] (1min4.2%vs 11.5%; 5min 2.1% vs 6.3%) A.M. Rathore *et al* [44]. (1 min 2% vs. 8%; 5 min 1% vs. 2%) reported similar findings in their studies. The Studies by Wenstrom[8] and Parson, Marci *et al*[19], Keith and Rogers all showed a significant decrease in the number of babies with low Apgar in the amnioinfusion group. This is in contrast with the study of Usta *et al*[27], which showed no significant difference between the Apgar scores of the two groups.

There was no significant difference in the birth weight of the babies from the amnioinfusion and control groups (2.77 vs.2.78). In the study by Usta *et al*[27] the mean birth weight was significantly higher in the amnioinfusion group.

In this study the presence of meconium below the level of vocal cord is significantly reduced in the amnioinfusion group (20% vs. 36% P<. 05). The incidence in other studies is given in Table 19. All the studies show a reduction in the incidence of meconium below the level of vocal cords with amnioinfusion except that of Sponge *et al*[26] Usta *et al*[27]

The incidence of meconium aspiration syndrome in this study was 6 in the amnioinfusion group versus 18 in the control group. (P<.05). This shows that there is significant decrease in the incidence of meconium aspiration syndrome in the group that received amnioinfusion. Other studies showing similar results are given in table 20. All the studies show decrease in the incidence of meconium aspiration syndrome with amnioinfusion.

TABLE 19

INTRAPARTUM AMNIOINFUSION AND PRESENCE OF MECONIUM BELOW THE VOCAL CORDS

Study	Amnioinfusion Group n (%)	Control Group n (%)
Sadovsky et al 1989	0/19 (0.0)	6/21 (28.6)
Macri et al 1992	4/85 (4.7)	33/85 (38.8)
Spong et al 1994	3/43 (7.0)	2/50 (4.0)
Cialone et al 1994	2/47 (4.2)	34/58 (58.6)
Eriksson et al 1994	1/65 (1.5)	8/59 (13.6)
Usta et al 1995	28/97 (29.0)	31/112 (28.0)
Hofmeyr et al 1998	6/158 (3.8)	12/164 (7.3)
Khosla et al 1997	3/25 (12.0)	4/25 (16.0)
A.M Rathor et al 2002	10/100 (10.0)	24/100 (24.0)
Sood M et al 2004	17/100 (17.0)	48/100 (48.0)
Parthe Mukhopadhyay et al 2006	6/93 (6.3)	34/93 (35.76)

TABLE 20

INTRAPARTUM AMNIOINFUSION AND MECONIUM ASPIRATION SYNDROME

Study	Amnioinfusion Group n(%)	Control Group n (%)
Wenstrom et al 1989	0/36 (0.0)	3/44 (6.8)
Macri et al 1992	0/85 (0.0)	5/85 (5.9)
Lo and Rogers et al 1993	1/60(1.7)	3/52 (5.8)
Cialone et al 1994	1/47 (2.1)	6/58 (13.6)
Erikson et al 1994	0/65 (0.0)	2/59 (3.4)
Usta et al 1995	19/497 (3.8)	20/440 (4.5)
Mahomed et al 1998	10/323 (3.1)	42/329 (12.8)
A.M. Rathor et al 2002	0/100 (0)	1/100 (1.0)
Partha Mukhopadhyay et al 2006	2/100 (2)	9/100 (9)
Das AK et al 2007	4/100 (4)	18/100 (18)

In this study 20 neonates from the amnioinfusion group and 38 from the control group needed admission in the neonatal unit. This shows that there is a significant decrease in the neonatal admissions in the amnioinfusion group. (20% vs. 38% P<. 05)

CRAMP 2 Zimbabwe arm[35] showed similar results (12.8% vs.22.9%). The study by A.M.Rathore *et al*[44] also showed decreased neonatal admissions in the amnioinfusion group. (3% vs. 11%) Studies by Eriksson *et al*[22] Cialone *et al*[23] and CRAMP 1 South African arm[34] also showed similar results.

In this study Hypoxic ischaemic encephalopathy occurred in 1 baby from each group. In the study by Moodley *et al*[36] hypoxic ischaemic encephalopathy occurred in none of the babies from the amnioinfusion group while 2 babies in the control group had neonatal hypoxic ischaemic encephalopathy.

The duration of stay in the neonatal unit was not significantly different in both the groups. This is similar to the study by Sponge et al 3.5 days in the amnioinfusion group versus 3.6 days in the control group. In this study the mean duration of stay in the neonatal unit was 3.2 days in the amnioinfusion group and 3.6 days in the control group.

The perinatal mortality rate in the amnioinfusion group is 1% while in the control group it is 3%. The difference is not significant statistically. In the CRAMP 2 study[35] conducted in Zimbabwe, the perinatal mortality was 1.2% in the amnioinfusion group and 3.6% in the control group. In the study by A.M. Rathore *et al*[44]. the perinatal mortality rate was 2% in the amnioinfusion group versus 5% in the control group. Das AK *et al* showed a perinatal mortality rate of 4% in the amnioinfusion group and 13% in the control group.

There were no increased puerperal complications in the amnioinfusion group. The incidence of puerperal pyrexia was 2% in the amnioinfusion group and 6% in the control group. This reduction was not statistically significant. In the study by A.M.Rathore *et al*[44] there was significantly reduced incidence of puerperal pyrexia in the amnioinfusion group. (6% vs. 12%) The studies by Wenstrom *et al*[8],

Sadovsky *et al*[9], Marci *et al*[19], have showed no increase in the maternal morbidity in the amnioinfusion group.

In this study no adverse effect was noted in the fetus or mother due to amnioinfusion.

SUMMARY

200 patients with moderate or thick meconium stained amniotic fluid were selected from labour ward in this prospective case controlled study. 100 of them were allotted to the amnioinfusion group and 100 were kept as control and received no amnioinfusion.

In analyzing the intrapartum course and the perinatal outcome, the following points were summed up.

1) Majority of patients in the amnioinfusion and control group were in the age group of 18-30. Only 6% in the amnioinfusion and 5% in the control group were above 30.

2) In both groups nullipara were predominant and contributed to 65%of the selected sample group in amnioinfusion and 61% of the selected sample group in control group.

3) The gestational age for majority of the patients in both the amnioinfusion and control group was between 37-40 weeks. Only 35% in the amnioinfusion group and 31% from the control group had gestational age above 40 weeks.

4) At the time of inclusion, majority of the patients were in the active phase of labour - 64% in the amnioinfusion group and 69% in the control group.

5) Augmentation was needed in 37% patients in the amnioinfusion group and 31%

patients in the control group.

6) The mean duration of labour from rupture of membranes to delivery was 1.73 hours in the amnioinfusion group and 1.56 hours in the control group. This difference is not significant and this shows that in this study there was no effect on the duration of labour by amnioinfusion.

7) The incidence of intrapartum fetal heart rate abnormalities was significantly lower in the amnioinfusion group.28% versus 42%.

8) Regarding the mode of delivery majority in both groups delivered by labour natural - 55% in the amnioinfusion group and 54% in the control group. The incidence of operative vaginal deliveries was 8% in the amnioinfusion group and 7% in the control group.

9) The number of patients who delivered by cesarean section was 37% in the amnioinfusion group and 39% in the control group. This difference was not significant.

10) There was significant decrease in the number of cesareans done for fetal distress in the amnioinfusion group. 18% versus 31% in the control group.

11) Most of the patients who were included in this study in the active phase of labour delivered vaginally while most of the patients who were included in the latent phase underwent cesarean section.

12) On summing up the data about the perinatal outcome, there was significant

decrease in the incidence of 1 minute and 5 minute Apgar scores less than 7 in the amnioinfusion group. 1 minute Apgar 41% in the amnioinfusion versus 60% in the control; 5 minute Apgar 13% in the amnioinfusion versus 24% in the control.

13) The mean weight of the babies was 2.77 in the amnioinfusion group and 2.78 in the control group.

14) The presence of meconium below the vocal cords was significantly reduced in the amnioinfusion group 20% versus 36% in the control group.

15) The incidence of meconium aspiration syndrome was also significantly reduced in the amnioinfusion group. 6% versus 18% in the control group. This shows that amnioinfusion is effective in reducing meconium aspiration syndrome in cases of meconium stained amniotic fluid.

16) Number of babies admitted to the neonatal unit was 20% from the amnioinfusion group and 38% from the control group. There is significant reduction in the neonatal admission with the use of amnioinfusion.

17) There was no significant difference in the duration of neonatal unit stay between the two groups. 3.2 days in the amnioinfusion group and 3.6 days in the control group.

18) Perinatal mortality was one in the amnioinfusion group and three in the control

group.

19) There were no increased puerperal complications in women who received amnioinfusion - 2% in the amnioinfusion group and 6% from the control group had puerperal pyrexia.

20) There were no adverse maternal or fetal complications reported with amnioinfusion in this study.

CONCLUSION

In this study it has been found that the use of amnioinfusion has significantly reduced the number of intrapartum fetal heart abnormalities.

It is not affecting the mode of delivery in patients with meconium stained amniotic fluid in labour. The incidence of cesarean delivery is not reduced but the number of cesareans done for fetal distress has decreased by the use of amnioinfusion.

The results show a striking improvement in the perinatal outcome in the patients who received amnioinfusion. There was significant improvement in the 1 minute and 5 minute Apgar scores, decrease in the incidence of meconium detected below the level of vocal cords and decrease in meconium aspiration syndrome. The number of babies admitted in the neonatal unit is also significantly reduced in the amnioinfusion group.

In the mother there was no complications related to amnioinfusion and there was no significant difference in the puerperium.

To conclude amnioinfusion is a safe, easy and effective procedure that needs little expertise and can be done in labour wards with even limited facilities. The use of such simple technique will not only reduce the burden on already limited resources in developing countries but will also have cost benefits.

ABBREVIATIONS

LN	-	Labour Natural
LSCS	-	Lower Segment cesarean section
LMC Forceps	-	Low mid cavity forceps
FD	-	Fetal Distress
NICU	-	Neonatal Intensive Care Unit
MAS	-	Meconium Aspiration Syndrome
HIE	-	Hypoxic Ischaemic encephalopathy
BA	-	Birth Asphyxia

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PROFORMA

Case Number :

Name : Age : I.P. No.

Obstetric Formula :

Menstrual Cycles : Regular / Irregular

LMP : EDD : GA Weeks :

Height: Weight :

PR : BP : Pallor :

Pedal Edema : CVS : RS :

P/A Uterine Size: Cephalic : Acting / not acting :

Fetal Heart Rate :

Rupture of membranes: date : time :

P/V cervical effacement dilatation : Station :

Pelvis : Degree of meconium :

Time of start of amnioinfusion :

Oxytocin augmentation : Yes / No

Intrapartum fetal distress : Yes / No

Delivery : Date : Time :

Mode of delivery : Natural/LSCS/LMC/Outlet Forceps

Indication for LSCS / Forceps : Fetal Distress / others

Rupture of membranes to delivery interval:

Baby : Alive / asphyxiated / dead born Sex : M / F

Apgar 1 min 5 min Baby Weight

Meconium below the vocal cord: Yes / No

Admission to NICU : Yes / No

Reason for Admission : Observation / MAS / BA / HIE

Duration of NICU Stay :

Condition on discharge :

Maternal Complications :